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ER Stress Mediated Apoptosis †

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Abstract: Endoplasmic reticulum (ER) is the major site where lipid synthesis, protein synthesis- its proper folding, maturation, and transport to different sites of cells, tissues, and organs occur. ER also is the storehouse of calcium ions required for various functions of the cells. Hence, for the proper maintenance of ER homeostasis, tight regulation of ER functions is essential. Many factors contribute to the misfolding of proteins in the ER lumen. The accumulation of these misfolded protein causes the ER to malfunction. Due to the misfolded protein stress, the ER malfunction initiates an adaptive response in the cell, unfolded protein response (UPR). PERK, IRE1, ATF6 are the key players in the initiation and regulation of UPR. These ER sensors induce ER-localized chaperones to downregulate protein synthesis and cause upregulation of the degrading protein system. However, if this mechanism fails to alleviate stress in the ER, this system then initiates apoptosis. This review focuses on how the complex role of ER and its signaling pathways provides a novel angle on apoptosis research and newly outlined signaling pathways of UPR.

Keywords: ER stress; UPR; apoptosis; PERK; IRE1; ATF6.

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Conflicts of Interest

The authors declare no conflict of interest.